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Fred E. Guedry, Jr., et al

Naval Aerospace Medical Research Laboratory

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Fred E. Guedry, Jr., Richard D. Gilson, David J. Schroeder, and William E. Collins

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Approved by

Ashton Graybiel, M.D.
Assistant for Scientific Programs

Released by

Captain N. W. Allebach, MC, USN
Officer in Charge

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NAVAL AEROSPACE MEDICAL RESEARCH LABORATORY
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13. ABSTRACT

Recent studies have shown that alcohol interferes with visual control of vestibular nystagmus. The present study was designed to assess three partially independent systems of oculomotor control. Performance on three tasks was measured before and after mild alcohol dosage. One task involved visual suppression of vestibular nystagmus; a second involved smooth oculomotor tracking of a moving target; and a third required repetitive rapid voluntary shifts in gaze. Oculomotor control was degraded on the first two tasks with recovery toward the initial performance level 4 hours after drinking. Performance on the third task was not obviously degraded, although it is possible that improvement with practice was retarded. Results are discussed in terms of neurological systems involved and kinds of flight tasks potentially affected.

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SUMMARY PAGE

THE PROBLEM

Previous observations showed that consumption of small amounts of alcohol, insufficient to influence control of a flight instrument in a stationary situation, caused significant performance degradation when the man was in motion. This was attributable to reduced visual override of the reflexive vestibular control of eye movements. The present experiment, by including several tasks which emphasize different aspects of oculomotor control, seeks to estimate the relative effects of alcohol on partially independent control systems to provide a basis for estimating the kinds of visual tasks which might be disturbed in flight.

FINDINGS

Performance on three oculomotor control tasks, one which emphasized visual suppression of vestibular nystagmus, another which required smooth oculomotor tracking of a moving target, and a third which demanded repetitive, rapid, voluntary shifts in gaze, was studied before and at 1, 2, and 4 hours after alcohol consumption. Eye movements were recorded and measured in all three tasks. In the first two tasks, ability to control a flight instrument was also measured. One hour after drinking, performance and oculomotor control were degraded on Tasks 1 and 2; some recovery was evident 2 hours after drinking; and oculomotor and tracking performance were almost to the initial level 4 hours after drinking on these two tasks. There was no apparent degradation in the third task, although it is possible that improvement as a result of practice was retarded. It is suggested that the same physiological mechanism may account for the effects on the first two tasks. However, the present results leave open the possibility that moderate alcohol consumption may have some influence on the voluntary saccadic system as well as on the other oculomotor control systems evaluated. In any case, it appears that alcohol will degrade performance on any tasks in which relative motion between the eye and the target approaches the limits of normal oculomotor control, irrespective of whether the relative motion is generated by vestibular control of the eye or by motion of the target.

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Dr. Guedry is associated with the Naval Aerospace Medical Research Laboratory; Dr. Gilson with the Department of Aviation, The Ohio State University, Columbus, Ohio; Dr. Schroeder with the V. A. Hospital, Murfreesboro, Tennessee; and Dr. Collins with the FAA Civil Aeromedical Institute, Oklahoma City, Oklahoma.

INTRODUCTION

A visible target fixed relative to the head substantially suppresses vestibular nystagmus, but this suppression is reduced by moderate doses of alcohol. Because vestibular nystagmus in the dark is relatively unaffected by comparable alcohol dosage (8), it appears to be the visual-vestibular interaction which is in some way changed. However, reduced optokinetic following has been reported following alcohol ingestion (1,15), and both effects, i.e., changes in optokinetic tracking and in visual-vestibular interactions, may be attributable to a common mechanism involving the use of retinal error signals, even though static visual acuity is regarded as insensitive to the effects of alcohol (20).

There are several systems of oculomotor control which involve some of the same final common mechanisms, but which differ in that each involves neurological systems not integral to the others (14). It is known, for example, that voluntary saccadic eye movements can be influenced by central nervous system lesions which leave smooth oculomotor tracking unaffected, and the converse is true of other lesions. Vestibular nystagmus may be normal with disorders which alter visually elicited eye movement. There is diagnostic significance in comparing vestibular nystagmus with and without visual fixation stimuli (10). It is reasonable, therefore, to study the influence of alcohol on different forms of oculomotor control.

The practical nature of this line of inquiry for aviation was indicated by previous observations showing that an alcohol dose, insufficient to produce a significant change in a simple task involving control of a flight instrument in a static situation, caused significant performance degradation when the man was in motion (8). The present experiment consists of an evaluation of the relative effects of alcohol on three tasks: one which emphasized visual suppression of vestibular nystagmus; another which required smooth visual tracking of a target; and a third which demanded repetitive rapid voluntary shifts in gaze. The set of tasks, by sampling different aspects of oculomotor control, provides a basis for estimating which kinds of visual oculomotor tasks might be disturbed in the flight environment as a result of moderate alcohol consumption.

METHOD

SUBJECTS

Eleven young naval and marine officers served as subjects. All were engaged in training and duties involving flying, and each had 20/20 vision or correction to 20/20.

APPARATUS

A detailed description of the rotational apparatus has been given elsewhere (8,17). The Human Disorientation Device (HDD) provided a sinusoidal whole-body motion about an Earth-vertical axis. The subject was seated upright in the HDD capsule with his head close to the axis of rotation and positioned to place the horizontal semicircular canals in

the plane of rotation. Sinusoidal rotation about an Earth-vertical axis with a frequency of 0.04 Hz and a peak angular velocity of ±60 deg/sec was the vestibular stimulus. The compensatory tracking task, previously described by Gilson et al. (8), consisted of maintaining the vertical needle of a cross-pointer (ILS) aircraft instrument in null position by manipulating a joystick to override a forcing function. Deviations of the needle from the null vertical position were considered errors which were electronically rendered in absolute values and integrated over consecutive 1-second intervals for 50 seconds or two complete cycles of the HDD. The display was positioned 0.8 meters in front of the subject. With the HDD interior otherwise darkened, localized illumination of the instrument display was provided by a small projector to give pointer luminance of 0.1 ft-L.

The same compensatory tracking task was also performed by the subject with a duplicate display in motion while he remained stationary. In this case, effective movement was produced by sinusoidal oscillation of a mirror which moved the reflected image to the left and right of the subject's line of regard. The display image moved at a frequency of 1.0 Hz with a visual angle between the two extreme positions of 10 degrees and a peak angular velocity of 31 deg/sec. Details of the apparatus are described elsewhere (7). The subject peered into an enclosure surrounding the equipment that allowed only the display face to be illuminated. Luminance as before was maintained at 0.1 ft-L and the viewing distance at 0.8 meters. The 50-second tracking period was retained although it was always preceded by an additional 15 seconds of unscored tracking to provide some accommodation to the task.

PROCEDURES

The experiment consisted of four testing sessions, an initial session and three post-drinking sessions 1, 2, and 4 hours after ingestion of alcohol. Each testing session consisted of the following sequence:

A. With the HDD stationary, brief practice on the compensatory tracking task in the HDD was followed by 1 minute of scored tracking. Oscillation of the HDD then commenced with subjects in complete darkness to obtain records of horizontal nystagmus without visual suppression. Eye movements were recorded by amplifying corneoretinal potential picked up by electrodes at the outer canthi. With the HDD still oscillating, the display was illuminated and the tracking task was resumed for 2.5 minutes.

B. Subjects were then moved to another experimental area where the compensatory tracking task was again performed for 50 seconds with the display stationary and then for 50 seconds with the image of the display in motion by virtue of oscillation of the viewing mirror.

C. Measures were taken of the maximum rate of voluntary horizontal shifts in visual fixation back and forth between two constantly illuminated spots separated by a visual angle of 20 degrees. These measures were obtained over two 20-second testing periods before and after the mirror tracking test. An identical test was run for vertical excursions.

Following this series of tests in the initial session, each subject consumed 2 ml of 100-proof Smirnoff vodka per kg of body weight in a mixture with 900 ml of orange juice in a period of 30 minutes. The same sequence of tests was repeated in each of the postdrinking sessions. Venous blood samples were drawn prior to each testing session, including the initial session, to permit determination of blood alcohol percentages by gas chromatography.

RESULTS

BLOOD ETHANOL LEVELS

There was no sign of ethanol in the predrinking blood samples, and the mean blood ethanol levels at the 1-, 2-, and 4-hour test sessions were .081 percent, .075 percent, and .047 percent, respectively.

OCULOMOTOR CONTROL

The mean alteration in slow phase velocity of nystagmic eye movements during performance of the tracking task in the HDD (Test A) is shown in Figure 1. The present results confirm previous reports of substantial degradation of visual suppression of vestibular nystagmus by mild alcohol intoxication (8).

There was also a change, following the alcohol dose, in the smooth sinusoidal ocular tracking of the mirror reflection of the display (Test B). The amplitude of the eye movement was significantly reduced from the initial session to the first postdrinking session and then recovered toward the level of the initial response amplitude in the second and third postdrinking sessions, as shown in Figure 2. These shifts in amplitude between the initial and first postdrinking session and between the first and third postdrinking sessions were statistically significant ($P < .01$). There was also a change in the form of the eye movements. In the initial and final sessions, the eye movements were fairly smooth and sinusoidal, especially after initial practice, with occasional saccades interrupting the smooth following. However, in the first postdrinking session, saccades were more common and the eye movement waveform sometimes had the appearance of a compromise between a sinusoid and square wave as shown in Figure 3, more or less like the unpracticed oculomotor tracking reported by Stroud (18). Thus fairly smooth ocular tracking tended to be replaced by saccades of less than full excursion with increased fixation time at the extremes. As illustrated in Figure 3, the quality of the oculomotor tracking before and after alcohol was subject to individual differences. The shape of the response waveform in the first postdrinking session caused estimates of stimulus-response phase angle to be difficult, but there was no obvious shift in this aspect of the response from one session to the next.

Alcohol did not reduce the number or apparent quality of voluntary visual excursions back and forth between the two fixation points (Test C) for either horizontal or vertical excursions. There was no change in the time required to make ten cycles from the predrinking session to the first postdrinking session, and in the last two sessions, there

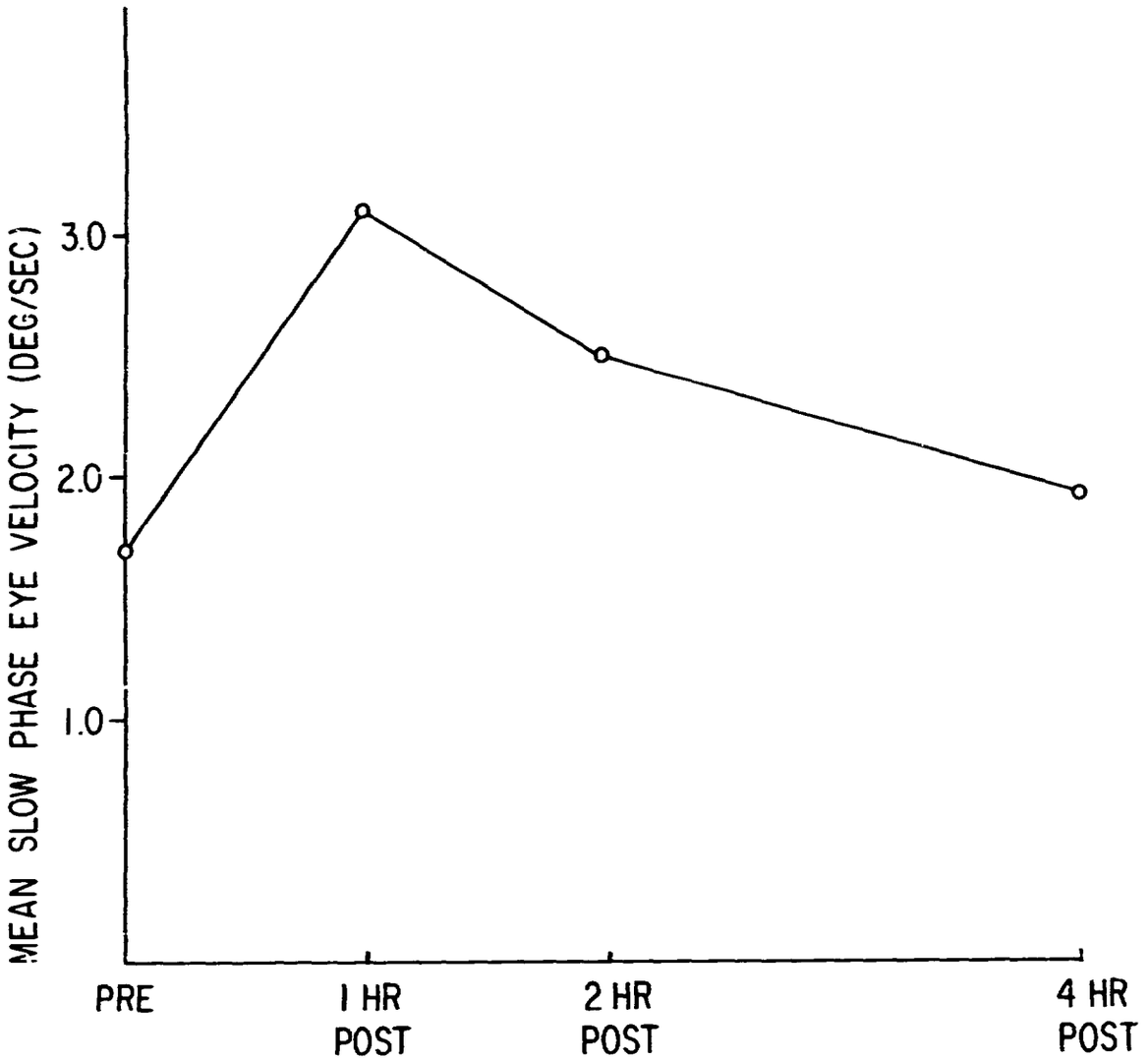

Figure 1

Mean slow phase velocity of visually suppressed nystagmus averaged over two cycles of the .04 Hz sinusoidal vestibular stimulus

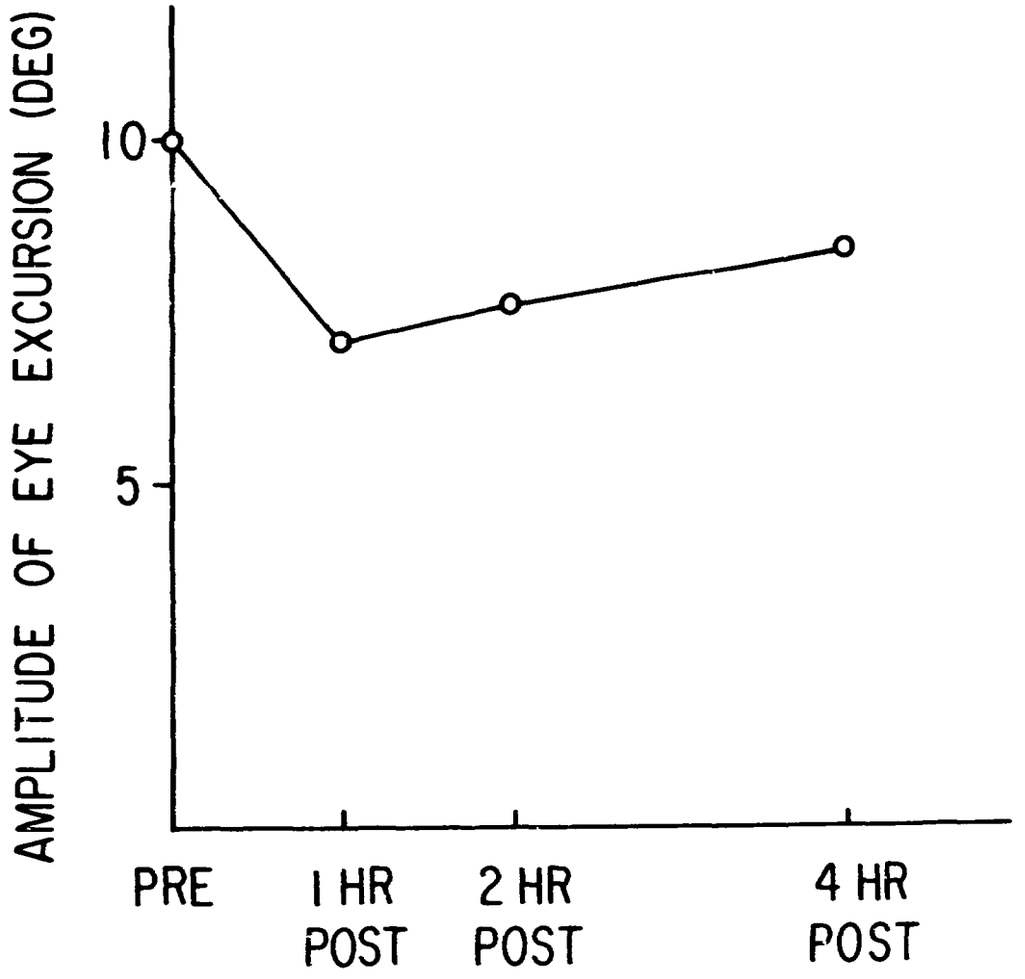

Figure 2

Amplitude of horizontal oculomotor tracking of an image moving with a peak-to-peak amplitude of 10 deg at a frequency of 1 Hz before and at three intervals after drinking alcohol

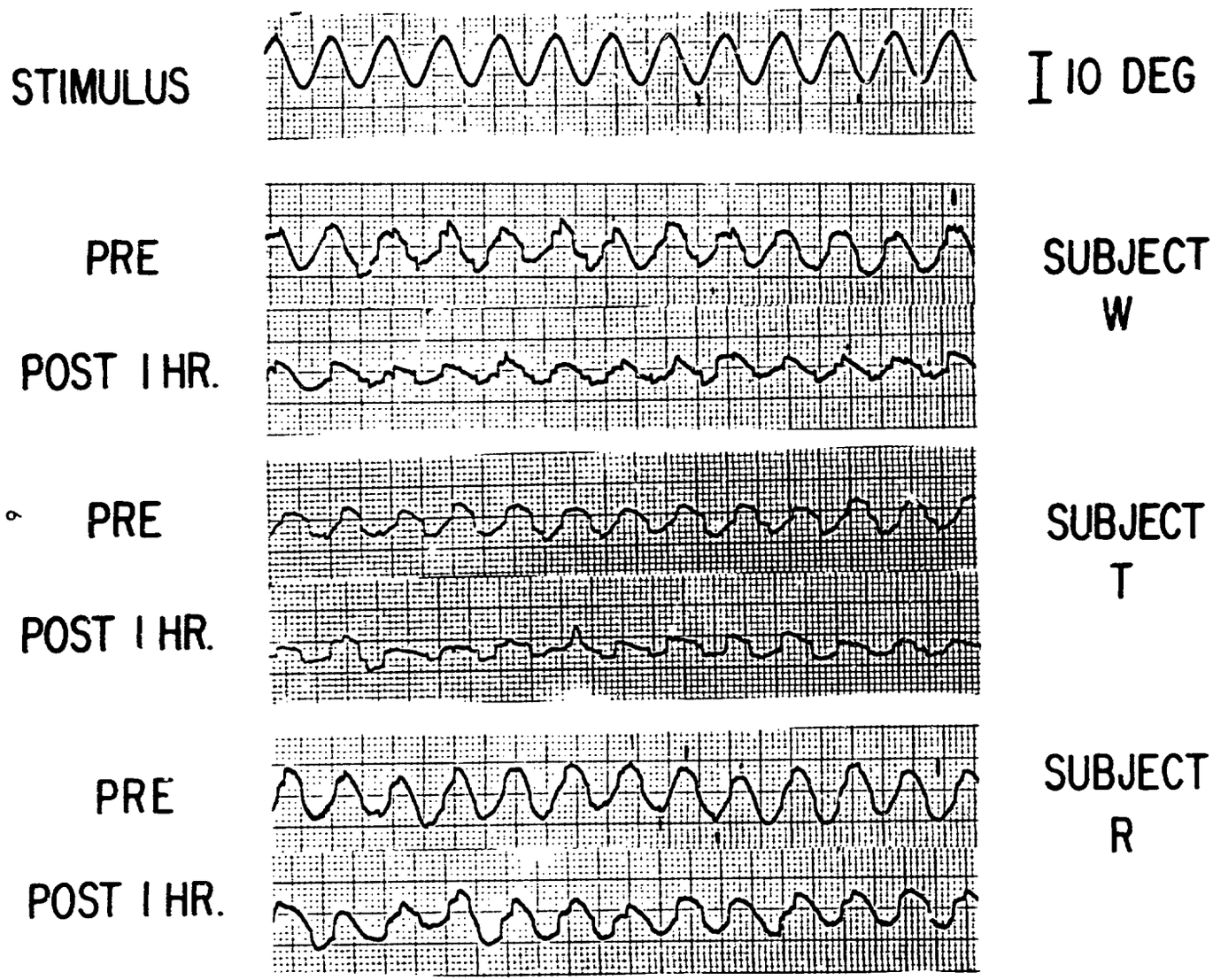

Figure 3

Samples of oculomotor tracking of mirror image before and 1 hr after alcohol consumption

was a slight increase in the number of to and fro excursions per 10-second interval (Figure 4). Since there was a significant increase ($P < .01$) in the number of excursions in the post-2-hour and post-4-hour sessions, it appears likely that alcohol did prevent this increase in the post-1-hour session.* The mean saccadic eye velocity was 413 deg/sec in the predrinking session, and in the post-1-hour session it was 359 deg/sec. This seems to confirm the findings of Franck and Kuhlo (6) who reported a slowing of voluntary saccades with alcohol intoxication. However, the mean velocity 4 hours after drinking was 364 deg/sec, nearly the same as the value obtained 1 hour after drinking. The mean blood ethanol level after 4 hours was .047 percent. The lowered velocity may have been due to a lowered visual attention (cf. 3) which in turn may have been attributable to either the remaining blood alcohol or some form of adjustment to the repeated testing. The fact that time to complete ten cycles actually decreased, i.e., cyclic frequency increased, while there was a decrease in saccadic velocity indicates that dwell time at the fixation point diminished. Perhaps slightly lower saccadic velocities are functionally mated to lower dwell times in a way which permits higher cyclic frequencies. Further experimentation will be required to answer such questions.

VISUAL PERFORMANCE

The tracking tasks in which the joystick was used to stabilize the cross-pointer served to maintain the interest and alertness of the subjects and in addition afforded a secondary measure of eye movement control. It should be noted that this compensatory tracking task does not demand a high level of visual acuity, but with sufficient image velocity caused by eye movement relative to the visual display, performance on the tracking task can be degraded.

This occurred in the present study as shown in Figure 5. The increase in error from the static to the dynamic condition, irrespective of alcohol, was considerably greater during mirror oscillation than during whole-body oscillation. However, the absolute increase in performance error was about the same when the post-1-hour session is compared with the predrinking session for either of two dynamic situations.

DISCUSSION

The effects of alcohol on both oculomotor control and the performance task were roughly the same during the visual-vestibular interaction and the visually guided tracking task. During oscillation on the HDD, the display was fixed relative to the display. To the extent that the slow phase of nystagmus occurs, there is an image velocity "error

*Time for the first ten complete cycles was used as the measure of performance in this analysis. All except one subject completed at least twenty full cycles in the time allotted before and after drinking alcohol. The eye movements of the exceptional subject became erratic and of lower frequency after the first ten cycles. When this subject is omitted and an analysis of variance for repeated measures is used on measures of time required for twenty full cycles, there is still a statistically significant difference ($P < .01$) between trials. With this deviant subject included by extrapolating to estimate the time he would have required for twenty cycles, the between-trials difference is questionable ($P < .10$).

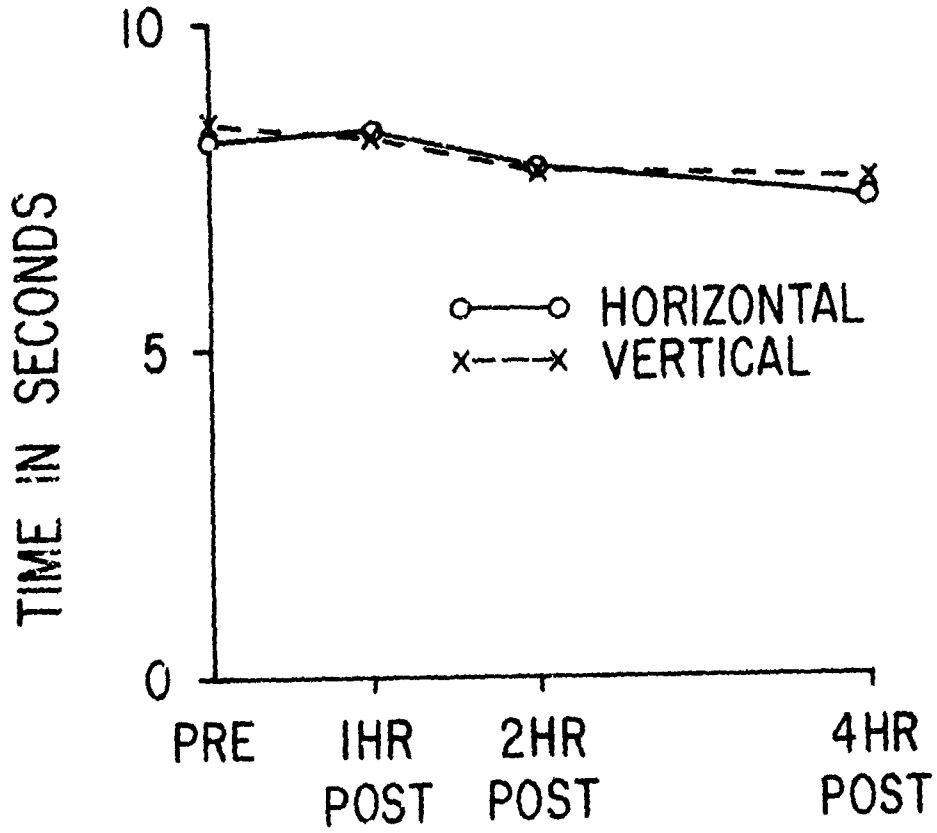

Figure 4

Mean times for ten cycles (20 saccades) of excursions between markers separated by 20-deg visual angles

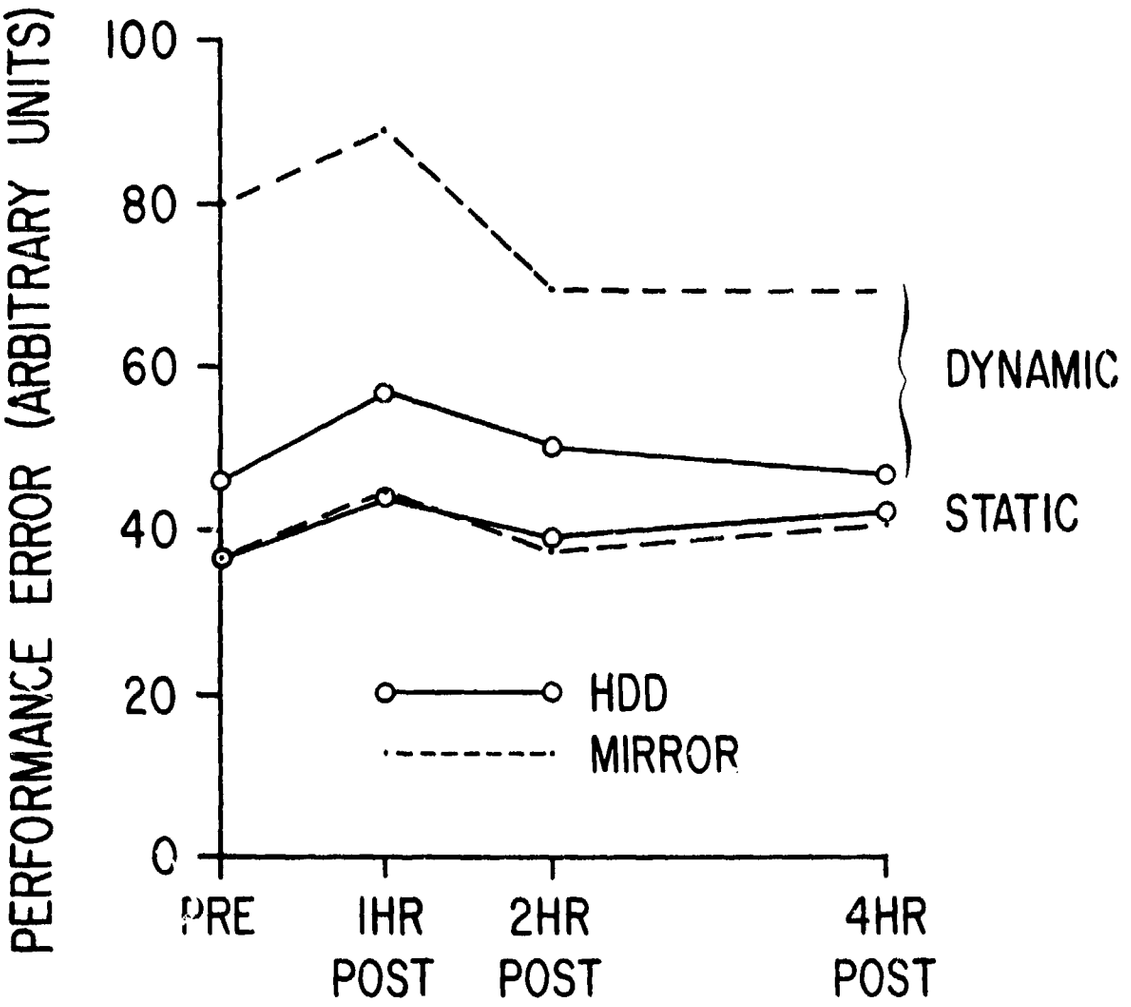

Figure 5

Performance measures in arbitrary units before and at three intervals after alcohol consumption

comparable to the "errors" which occur when optokinetic tracking does not match the angular velocity of a moving target. If the peak slow phase velocity of the eye in darkness during the vestibular stimulus is taken as the amount of error to be overcome by the visual fixation system, then the vestibular stimulus created a potential mean maximum mismatch between eye and display velocities of 40 deg/sec,* which varied from zero mismatch to the maximum of 40 deg/sec mismatch twice each cycle, i.e., at a frequency of .08 Hz. In contrast, although the peak velocity of the mirror image was only about 31 deg/sec, the relatively high frequency (1 Hz) oscillation created the potential of a more continuous velocity mismatch between the eye and the display than would occur during the lower frequency sinusoidal vestibular stimulation. Judging from the static to dynamic change in performance without alcohol, the visually guided tracking task was the more difficult. Even so, absolute changes in oculomotor control and in performance after alcohol were about the same in the two dynamic situations. Hence, it is possible that the loss of control in the two situations results from a common cause. It is possible that alcohol selectively disturbs centers responsible for reducing retinal slippage of images under selective attention, and this interfered with both the visual suppression of vestibular nystagmus and also the smooth pursuit tracking of the visual target. It will be necessary to achieve a closer match of the potential error signals created by these two different challenges to effective oculomotor control and performance to determine whether or not alcohol exerts a greater influence on one interaction than on the other, but no clear difference emerges from the present study.

The absence of degradation in the subject-paced task of executing rapid shifts in fixation suggests that the saccadic oculomotor system was not disturbed by the alcohol levels encountered. There was no obvious evidence of qualitative changes in these eye movements. This suggests that visual tasks requiring only fixation shifts would not be disturbed by moderate levels of alcohol intake, but to be sure of the relevance of this conclusion to aviation, it would be advisable to use a task requiring both rapid changes in fixation and quick information retrieval during the fixation (cf. 20, pp 300-301) and also to take into account that improvement as a result of practice seemed to be retarded. There was an increased cyclic frequency of these voluntary excursions in the second and fourth hours after alcohol, whereas there was no increase one hour after drinking. However, the lowering of saccadic eye velocities after alcohol ingestion emphasizes the need for a control group in further experimentation to clarify interpretation of these results. It is possible that lowered eye velocity permits shorter dwell time and that this functional combination develops with practice.

Effects on smooth pursuit tracking and on visual suppression of vestibular nystagmus, very similar to those noted in the present study, have been reported in connection with barbiturates (13,16), suggesting that some combinations of mild dosages of barbiturates and alcohol may produce effects even stronger than those noted in the present study. Because it has been reported that sleep deprivation increases the frequency of

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*The peak slow phase velocity of vestibular nystagmus for the same stimulus and subjects shown in a previous report (17) was about 20 deg/sec, but the calibration was in error by a factor of 2. This error was irrelevant to the main points in the previous paper, but it is inferentially relevant here.

nystagmus to fairly strong vestibular stimuli (22), combinations of sleep loss and mild alcohol dosage would be also an important area of inquiry in aviation. In regard to estimating categories of visual oculomotor control tasks which would be more influenced by the effects of alcohol, final conclusions cannot be recched from this study; but it appears that short-term performance in alert subjects is apt to be degraded in tasks which require either smooth visual tracking at high relative velocities or oculomotor control during vehicle oscillation (vestibular stimulation). Tasks which require only rapid shifts in visual fixation in a relatively stable environment may not be very much influenced, although this must be qualified by the apparent retardation of practice effects by alcohol. Hence, recently acquired skills in rapid instrument scan may be adversely influenced.

It has been suggested that the superior colliculus, which receives input directly from the retina and from the visual cortex is important in visually guided eye movements (14). However, Wurtz and Goldberg (23), noting that monkeys without superior colliculi did not exhibit gross deficits in visually guided eye movements, argue that the superior colliculi mediate shifts in attention which must precede displacements of the eye during visually guided movement. However, if this were the problem encountered in the present experiment following alcohol ingestion, then longer dwell time (and not just a retarded practice effect) might be expected in Task C, since Wurtz and Goldberg indicated that discrete lesions in the superior colliculus increased the latency but did not affect the accuracy of saccades to stimuli in the area affected by the lesions. Part of the effects on oculomotor control found in the present study may be related to an influence of alcohol on cerebellar function, but in view of the many sites of visual-vestibular interaction (cf. 11), this is only speculation. Visual suppression of vestibular nystagmus is reduced by lesions of the flocculus in primates (2,19) and the nodulus has been implicated in suppression of vestibular nystagmus (5). Possibly, then, the reduced visual suppression of vestibular nystagmus with mild alcohol dosage involves the flocculonodular lobe of the cerebellum. However, according to Kornhuber (9), the interference with smooth pursuit eye movements is not suggestive of cerebellar dysfunction but rather one would expect some hypometria in the voluntary saccadic excursions. None was found. If the cerebellum plays a major role in automatizing repeated voluntary actions (cf. 12), then perhaps the absence of practice effects in the frequency of saccadic excursions in the first postdrinking session may be attributable to temporarily disturbed cerebellar function. Although many of the effects of alcohol on the regulation of posture and movement resemble symptoms of cerebellar dysfunction, there is apparently a paucity of direct studies on the influence of alcohol on cerebellar function (4,20). The possibility that voluntary saccadic velocities were retarded by alcohol, though not definitely established by the present experiment, may be indicative of an effect on the reticular formation of the pons (cf. 3,21), so that the present results leave open the possibility that moderate alcohol consumption influences all of the systems of oculomotor control outlined by Robinson and others (14), albeit the smooth tracking system and visual-vestibular interaction may be more influenced, and the effects on both of these systems may be attributable to a single cause, viz., interference with the processing of retinal "error signals," perhaps velocity error signals.

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